



## Hypertensive crises: Opinion of the Kyrgyz society on hypertension (literature review)

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**Abstract.** Arterial hypertension is one of the leading causes of morbidity and mortality from cardiovascular diseases worldwide, including in the Kyrgyz Republic. One of the most serious complications of this disease is a hypertensive crisis. The urgency of revising current standards is dictated by the continuing practice of overdiagnosis of "uncomplicated" crises, which leads to unnecessary hospitalisations and potentially dangerous aggressive therapy. The purpose was to summarise and present contemporary approaches based on the principles of evidence-based medicine to the definition, classification, diagnosis, and treatment of hypertensive crises to introduce them into clinical practice in the Kyrgyz Republic. The methodology was based on a systematic search,

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analysis, and adaptation of key clinical guidelines from the European Society of Cardiology, the European Society of Hypertension, the International Society of Hypertension, the British and Irish Hypertension Society, the American College of Cardiology, the American Heart Association, and the Russian Society of Cardiology, published between 2018 and 2025, with particular attention to their applicability in resource-limited settings. The review presented a contemporary clinical definition of hypertensive crises based solely on the presence of acute damage to target organs. The rejection of the term “uncomplicated hypertensive crisis” as an indication for emergency intravenous pressure reduction was substantiated. The algorithm of the initial assessment (identification of “red flags of target organ damage”) was described in detail, and a minimum set of laboratory and instrumental studies was regulated. A differentiated treatment strategy was proposed depending on the type of target organ damage, indicating target blood pressure levels and priority parenteral medications. Special attention was paid to the risks of uncontrolled decrease in blood pressure in inpatient and outpatient settings, and tactics for reducing pressure with an asymptomatic increase. The introduction of uniform standards will reduce inappropriate hospitalisations, improve the safety of treatment, and coordinate tactics at all levels of medical care

**Keywords:** hypertensive emergencies; target organ damage; emergency care; clinical guidelines; arterial hypertension; malignant hypertension

### Introduction

Arterial hypertension (AH) remains one of the leading causes of cardiovascular morbidity and mortality both worldwide and in the Kyrgyz Republic (KR). One of the most severe complications of this disease is hypertensive crisis (HC), an emergency clinical condition that poses a direct threat to the patient’s life and requires urgent medical intervention. Despite currently available treatment options, such acute conditions continue to occupy a significant place in the structure of emergency cardiology. In the clinical practice of the Kyrgyz Republic, an objective assessment of the prevalence of HC is extremely difficult. The main reason for this is the systemic problems of differential diagnosis. On the one hand, the practice of overdiagnosis is widespread, when the diagnosis of a crisis is made solely based on high blood pressure (BP) in the absence of appropriate clinical symptoms, which leads to unjustified hospitalisations and potentially dangerous aggressive therapy. On the other hand, overdiagnosis is also observed, whereby developing acute target organ damage (TOD) is underestimated by clinicians because of a relatively modest elevation in blood pressure.

During the period from 2019 to 2024, international approaches to the HC terminology were radically revised [1-2]. In Kyrgyzstan, there is still a division of the HC into “complicated” and “uncomplicated” variants. International professional communities, including the European Society of Cardiology (ESC), the European Society of Hypertension (ESH), the International Society of Hypertension (ISH), the British and Irish Hypertension Society (BIHS), and the Russian Society of Cardiology (RSC) have reviewed approaches to the definition, classification, and management of hypertensive crisis, which has led to the need to analyse current clinical guidelines and their conceptual differences. In contemporary recommendations and position documents by G. Mancia *et al.* [3], T. Unger *et al.* [4] and S. Kulkarni *et al.* [5], HC is defined as a condition in which an increase in BC is associated with acute or progressive TOD,

including acute coronary syndrome (ACS), acute heart failure (AHF), aortic dissection, stroke, hypertensive encephalopathy, acute renal failure (ARF), and malignant hypertension with retinopathy and thrombotic microangiopathy.

The use of the term “uncomplicated hypertensive crisis” to refer to transient increases in BP without signs of acute TOD is currently considered inappropriate and excluded from most contemporary European recommendations, and documents of Z.D. Kobalava *et al.* [6], which is conditioned by the lack of convincing evidence of improved clinical outcomes with an urgent blood pressure reduction in this patient group; moreover, according to a meta-analysis by T.J. Siddiqi *et al.* [7], forced medical intervention is associated with the risk of hypoperfusion of vital organs.

The relevance of the revision of current standards in the Kyrgyz Republic is conditioned by a number of factors: firstly, according to Z.D. Kobalava *et al.* [6], the persistent overdiagnosis of HC in clinical practice, based mainly on BP levels without considering damage to target organs, leads to unnecessary hospitalisations and overly aggressive therapy; simultaneously, the clinical recommendations of J.W. McEvoy *et al.* [8] and S. Kulkarni *et al.* [5] demonstrated conceptual unity in rejecting emergency reduction of BP in asymptomatic cases. However, they differ in tactics and target blood pressure levels in the first hours in certain acute conditions; secondly, according to J.B. Miller *et al.* [2] and J.W. McEvoy *et al.* [8], the lack of unified terminology and classification limits the comparability of epidemiological data and disrupts the continuity of patient management at various stages of medical care.

This review presents the opinion of the Kyrgyz Society for Hypertension (KSH), aimed at introducing contemporary evidence-based approaches to the management of patients with HC, adapted to the healthcare opportunities of the Kyrgyz Republic. The main conclusions and recommendations of this position statement

are supported by the Association of Internal and Interdisciplinary Medicine (AIIM) of KR. The purpose of this study was to develop an adapted, scientifically based clinical algorithm for the management of patients with acute TOD on the background of AH to optimise standards of emergency medical care and reduce the frequency of unnecessary hospitalisations in the Kyrgyz Republic.

### Materials and Methods

The methodology of the present study comprised a narrative review with elements of a systematic literature search, critical analysis, and expert adaptation of clinical recommendations. The main task was not only the compilation of existing data, but also their clinical and pragmatic interpretation considering the specifics of the healthcare system of the Kyrgyz Republic. The current clinical recommendations of the leading European professional communities were used as fundamental sources: ESC [8], ESH [3] and BIHS [5]. The recommendations of international, American and Russian organisations were also followed: ISH [4], ACC/AHA [9], and RSC [6]. Additionally, the analysis included specialised consensus documents on the management of individual clinical scenarios of hypertensive crises (HC), including acute coronary syndrome, cerebrovascular events, and aortic lesion [10-12].

The information search was conducted in the PubMed/MEDLINE, Cochrane Library and eLibrary databases with a limited publication period (2019-2024), due to the need to analyse contemporary conceptual approaches to the definition and classification of HC. Combined search strategies using logical operators (AND, OR) and MeSH terms were used: “hypertensive emergency”, “hypertensive crisis”, “target organ damage”, “management”, “guidelines”, and their Russian-language counterparts. The search was complemented by a manual analysis of the literature lists of relevant publications. The inclusion criteria included the selection of full-text papers, systematic reviews, meta-analyses, randomised clinical trials, and official clinical recommendations. Publications with limited levels of evidence (case reports, small retrospective series), non-peer-reviewed materials, and duplicate datasets were excluded. The selection was carried out in two stages (screening of headings and annotations, followed by evaluation of full texts), considering the key principles of PRISMA, without formally constructing a systematic review protocol.

The quality of clinical guidelines was assessed using the AGREE II instrument, allowing consideration of methodological rigour and transparency of the presented recommendations. In the analysis of original studies, the class of recommendation and the level of evidence were considered. Special attention was given to the expert adaptation stage, aimed at translating international recommendations into the context of national clinical practice. The limitations associated with the availability

of medicines (in particular, the lack of registration of a number of parenteral antihypertensive drugs) and the variability of haemodynamic monitoring capabilities were addressed. In the context of the identified discrepancies between the recommendations of various professional communities, priority was given to more conservative strategies that minimise the risk of iatrogenic hypoperfusion.

Data extraction was performed using a standardised matrix and focused on the systematisation of therapeutic approaches that formed the basis of a differentiated management strategy. The following clinical parameters were extracted: recommended intensity (as a percentage of baseline) and rate of haemodynamic discharge, strict target blood pressure levels in the first hours of therapy, safety profiles of first-line drugs and alternative pharmacological agents in direct dependence on the identified clinical phenotype of acute target organ damage. The presented rigorous methodological approach made it possible to form a locally adapted, scientifically based algorithm for emergency care in HC, while maintaining adherence to the fundamental principles of evidence-based medicine.

### Definition, current concepts of pathophysiology, and clinical classification of hypertensive crises

HC is a condition in which a significant increase in BP is associated with acute TOD, which is often life-threatening and requires immediate qualified actions aimed at reducing BP, usually with the help of intravenous therapy. The outdated name of this condition is “complicated hypertensive crisis”. In English-language publications, the term “hypertensive emergencies” is used to define HC, characterising the importance of providing emergency care in this condition [1-2]. Traditionally, extremely high systolic (SBP > 200-220 mmHg) and diastolic blood pressure (DBP > 120-130 mmHg) values are recorded in HC. However, it is well known that there is no clear relationship between the clinical manifestations of HC and the value of BP: HC can occur in patients with a relatively small but individually high blood pressure, a sharp rise in which is sufficient to provoke organ failure [10-12]. Therefore, the key criterion for verifying the diagnosis and initiating emergency therapy is not the absolute values on the tonometer, but the markers of acute regional ischaemia (cerebral, cardiac, or renal) [3,8].

According to the recommendations of the Ministry of Health of the Kyrgyz Republic [13] and the current international clinical guidelines of G. Mancia *et al.* [3] and J.W. McEvoy *et al.* [8], when detecting a BP level corresponding to grade 3 AH, HC should be excluded. In this case, it is important to differentiate the concept of “HC” from poorly controlled, but asymptomatic and clinically stable increase in BP in patients with hypertension, the so-called “uncontrolled AH”. The statement

proposed by some RSC experts [6] to consider “uncontrolled AH” as “uncomplicated HC” is controversial, since these terms are not equivalent. The significance of this problem is particularly high in the Kyrgyz Republic: as noted by G.O. Narkulova *et al.* [14], insufficient control of AH at the outpatient stage inevitably leads to an increased incidence of acute cardiovascular complications. Currently, the vast majority of patients with hypertension have uncontrolled blood pressure; however, these patients do not meet the criteria for HC. They require planned treatment intensification and improved adherence to antihypertensive therapy, rather than emergency BP reduction.

It is also necessary to exclude a transient (often physiological) increase in BP (“situational AH”) against the background of pain, anxiety/stress reactions, physical exertion, and the effect of a “white coat” – previously, this condition was often classified as an “uncomplicated hypertensive crisis”, which led to an unjustified emergency and not always safe decrease in BP [7]. Uncontrolled AH and situational AH in some cases, with a sharp and pronounced acute increase in BP, can also lead to the development of clinical symptoms up to acute TOD – in this case, these conditions can be considered as causative factors of HC [1-2].

The “secondary” increase in BP in acute emergencies deserves special attention. Severe AH may be a concomitant symptom rather than a direct cause of the development of an emergency condition, and an emergency reduction in BP in this situation is not always justified and useful, according to B.H. van den Born *et al.* [1] and T.J. Siddiqi *et al.* [7]. Admittedly, the presence of acute TOD in AH is a prerequisite for the provision of emergency antihypertensive therapy; however, in clinical practice, the doctor needs to clearly differentiate the primary development of pathophysiological processes. Such acute emergencies as acute stroke, aortic dissection, and acute coronary syndrome are often associated with significant physiological stress and pain syndrome, which themselves may lead to a marked increase in blood pressure. The abrupt increase in blood pressure may act both as a cause of these emergency conditions and as their consequence; in the first scenario, urgent blood pressure reduction is beneficial, whereas in the second scenario, lowering blood pressure provides no clinical benefit [2]. Thus, it is advisable to specify the following criteria for HC: an acute increase in blood pressure directly causes acute TOD and its associated emergency; there is evidence that an immediate decrease in BP will bring clinical benefits. A typical example of hypertensive crisis is hypertensive encephalopathy; this condition meets the criteria described above, and patients require emergency BP reduction. Conversely, an urgent decrease in blood pressure in ischaemic stroke can be potentially dangerous [1].

The cause of HC is usually hypertension. HC can occur both *de novo* (in patients with previously

undiagnosed hypertension) and in patients with untreated and/or uncontrolled AH, and the latter is more common [2]. HC also develops in secondary forms of AH (vasorenal AH, diabetic nephropathy, diseases of the nervous system, pheochromocytoma, primary hyperaldosteronism, and in pregnant women (with the development of preeclampsia) and when taking drugs that can increase BP [14-16]. The pathophysiology of HC is a complex process of disruption of autoregulation of the vascular bed, closely related to cardiovascular remodelling [17], which leads to microcirculatory damage, vasoconstriction, and hypoperfusion of target organs [2].

In the recommendations of Z.D. Kobalava *et al.* [6], J.W. McEvoy *et al.* [8] and P.K. Whelton *et al.* [9], preference is given to the clinical classification of HC based on the severity of the clinical symptoms of acute or progressive TOD and the risk of developing life-threatening complications. The following classification of HC is proposed. Firstly, HC is isolated in patients with severe AH (usually grade 3) associated with conditions requiring careful BP monitoring [6,17-18]. These conditions include: acute cerebrovascular accident (haemorrhagic or ischaemic stroke), acute aortic aneurysm or dissection, acute heart failure, acute coronary syndrome, and acute renal failure. Secondly, malignant hypertension is classified as characterised by severe AH (grade 3 AH) with microvascular acute damage, usually to three or more target organs [2,19]. Manifestations of malignant hypertension include hypertensive retinopathy (retinal haemorrhages and/or optic disc oedema) [6], thrombotic microangiopathy/disseminated intravascular coagulation (DIC) [20], and hypertensive encephalopathy, characterised by impaired consciousness, cortical blindness, and seizures [21]. In addition, acute heart and kidney failure may develop with malignant hypertension [22]. Thirdly, the adrenergic HC associated with TOD is isolated, including an endogenous excess of catecholamines (pheochromocytoma/paraganglioma) [23] or an exogenous excess of catecholamines (substance abuse) [2]. Ultimately, HA is classified into a separate group for severe hypertensive disorders of pregnancy (preeclampsia/eclampsia) [24-25].

### **Algorithms of primary diagnosis and differentiated tactics of emergency antihypertensive therapy**

Diagnosis of HC is a process that requires a quick and accurate assessment of the condition of target organs in patients with suspected HC: the doctor must identify symptoms indicating acute TOD – the so-called “red flags of TOD”. These include chest pain, shortness of breath, intense headache, visual disturbances, neurological symptoms: seizures, impaired consciousness, cortical blindness and coma, focal symptoms, oliguria/anuria [1,26-27]. It is important to note that the absence of symptoms practically excludes the diagnosis of HC.

According to G. Grassi *et al.* [28], M.A. Talle *et al.* [29] and T.J. Wang *et al.* [30], the diagnostic algorithm for suspected HC includes several consecutive stages. The first stage is the correct measurement of blood pressure: it is necessary to confirm elevated BP with a series of measurements using a standardised technique in a calm environment, and exclude situational hypertension (as a reaction to pain, stress, etc.) [28-30]. The second step is obtaining the medical

history and performing a physical examination: it is necessary to clarify the presence of CVD in the history, adherence to therapy, precipitating factors, assess neurological status, the presence of pulmonary rales, oedema, and pulse asymmetry [1]. The final stage is to conduct laboratory and instrumental studies: the volume is determined by the clinical picture [1,31], but there is a mandatory minimum for patients with suspected HC [32-34] (Table 1).

**Table 1.** Examination of patients with suspected HC to exclude acute TOD

|                                                                                                                                                  |
|--------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Standard examination</b>                                                                                                                      |
| ECG (12 leads) – no later than 15 minutes after presentation                                                                                     |
| Performance of the following laboratory tests:                                                                                                   |
| ■ complete blood count (CBC) (assessment of red blood cells, platelets, leukocytes, and haematocrit levels in blood)                             |
| ■ measurement of serum potassium and sodium levels in blood                                                                                      |
| ■ measurement of serum creatinine levels and estimated glomerular filtration rate (eGFR) assessment                                              |
| ■ assessment of fibrinogen levels                                                                                                                |
| ■ assessment of albuminuria, albumin/creatinine ratio                                                                                            |
| ■ microscopy of urine sediment (erythrocytes, leukocytes, cylinders)                                                                             |
| <b>Special tests performed according to indications</b>                                                                                          |
| Measurement of haptoglobin levels and lactate dehydrogenase (LDH) activity (if available) in cases of suspected thrombotic microangiopathy (TMA) |
| TSH and f.T4 in cases of suspected thyroid pathology                                                                                             |
| Pregnancy test (in patients of reproductive age) for suspected preeclampsia/eclampsia                                                            |
| Determination of the level of troponin, MV fraction of creatine kinase in case of suspected ACS                                                  |
| Determination of the level of NT-proBNP in case of suspected heart failure                                                                       |
| Ophthalmoscopy (fundoscopy) for suspected malignant hypertension with retinopathy                                                                |
| Neurological examination in case of suspected ACVA                                                                                               |
| Chest X-ray to clarify the aetiology of dyspnea                                                                                                  |
| Echocardiography in case of suspected aortic dissection, OSN, ACS                                                                                |
| CT angiography of the chest and/or abdominal cavity in case of suspected acute aortic injury                                                     |
| CT or MRI scan of the brain in case of suspected ACVA                                                                                            |
| US of the kidneys in case of suspected renal artery stenosis or kidney damage                                                                    |
| Urine testing for narcotic substances in case of suspected use of methamphetamine or cocaine                                                     |

**Note:** ECG – electrocardiography, TSH – thyroid-stimulating hormone, f.T4 – free thyroxine, ACS – acute coronary syndrome, NT-proBNP – precursor of cerebral natriuretic peptide, ACVA – acute cerebrovascular accident, CT – computed tomography, MRI – magnetic resonance imaging, US – ultrasound examination

**Source:** developed by the authors based on a study by B.H. van den Born [1]

Based on the minimal set of laboratory and instrumental studies performed, the clinician can reliably exclude or confirm the presence of acute TOD, which is a determining factor for choosing further safe therapeutic tactics.

### Treatment of hypertensive crises

The main task of medical relief of HC is a smooth, strictly controlled decrease in BP. Therapy is aimed at minimising further tissue damage while maintaining adequate perfusion pressure in the myocardium, kidneys, and, most importantly, in the brain, since cerebral blood flow is most vulnerable to sudden haemodynamic changes. As claimed by J.W. McEvoy *et al.* [8], HC treatment should be differentiated depending on the presence, nature, and severity of the target organ damage. Studies by B.H. van den Born *et al.* [1], Z.D. Kobalava *et al.* [6] and J.W. McEvoy *et al.* [8] confirmed that

patients with HC require emergency hospitalisation in the department of emergency cardiology or intensive care unit and require immediate qualified actions aimed at enhanced BP control using intravenous therapy. The choice of tactics for reducing or intensively controlling blood pressure will depend on the type of TOD, since not all types of acute TOD on the background of HC require an emergency reduction in BP (for example, with cerebral strokes, caution should be exercised, and in some cases, even antihypertensive therapy should be abandoned) [1,6,8].

The main provisions defining treatment tactics for HC include identifying the type of acute TOD to determine the need for any special intervention in addition to lowering BP, and identifying the causes of HC that may affect the choice of treatment tactics (for example, pregnancy). The timing, rate, and magnitude of a safe decrease in blood pressure should be determined: for

different variants of HC, the choice of intravenous anti-hypertensive drugs varies slightly, but the rate and intensity of BP reduction vary significantly; in most cases, the use of intravenous AHPs with a short half-life is indicated, which allows careful monitoring of BP response to treatment under constant haemodynamic monitoring to prevent organ hypoperfusion- targets. Careful

follow-up and long-term monitoring are also necessary: even with a successful rapid decrease in BP, insufficient attention is often paid to long-term correction of blood pressure and prevention of the progression of TOD. The recommended intensity of BP reduction and medications for various emergency clinical situations against the background of HC are shown in Table 2.

**Table 2.** Recommended intensity of BP reduction in HC and medications recommended for various emergency clinical situations in HC

| Clinical conditions                                                                            | BP reduction rate, target BP level                                                                                                                                                             | First line of treatment                                                  | Alternative AHP                                        |
|------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|--------------------------------------------------------------------------|--------------------------------------------------------|
| <b>Malignant AH, with or without acute renal failure<sup>†</sup></b> [1,12]                    | Decrease in avg. BP for several hours at 20-25%                                                                                                                                                | Labetalol*<br>Nicardipine*                                               | Sodium nitroprusside*<br>Urapidil                      |
| <b>Hypertensive encephalopathy</b> [1,12]                                                      | Immediate reduction in avg. BP by 20-25%                                                                                                                                                       | Labetalol*<br>Nicardipine*                                               | Sodium nitroprusside*<br>Urapidil                      |
| <b>Acute ischaemic stroke</b> [35-37]                                                          | Reperfusion is planned: reduce and maintain BP at a level of less than 185/110mmHg for at least 24 hours                                                                                       | Labetalol*<br>Nicardipine*                                               | Sodium nitroprusside*<br>Urapidil                      |
|                                                                                                | No reperfusion is planned:<br>■ with BP >220/120 mmHg: decrease in avg. BP for 15-20%<br>■ with blood pressure <220/120 mmHg: <b><i>routine reduction is not indicated within 72 hours</i></b> |                                                                          |                                                        |
| <b>Acute haemorrhagic stroke</b><br>■ less than 6 hours [38-40]<br>■ more than 6 hours [41-43] | Less than 6 hours: reduction to BP<140/90 (but not more than 60 mmHg from baseline)                                                                                                            | Labetalol*<br>Nicardipine*                                               | Sodium nitroprusside*<br>Urapidil                      |
|                                                                                                | More than 6 hours:<br>■ with SBP >220 mmHg, gradual decrease to SBP <180 mmHg<br>■ with SBP <220 mmHg, slow decrease to BP <140/90 mmHg                                                        |                                                                          |                                                        |
| <b>Acute coronary syndrome</b> [44]                                                            | Immediate reduction of SBP to <140 mmHg                                                                                                                                                        | Nitroglycerin<br>Labetalol*                                              | Urapidil<br>Esmolol*<br>Metoprolol*                    |
| <b>Acute cardiogenic pulmonary oedema/cardiac asthma</b> [45]                                  | Immediate reduction of SBP to <140 mmHg                                                                                                                                                        | Sodium nitroprusside*<br>Nitroglycerin<br>Loop Diuretics<br>Enalaprilat* | Urapidil                                               |
| <b>Acute aortic dissection</b> [46]                                                            | Immediate reduction of SBP to <120 mmHg and HR <60 bpm                                                                                                                                         | Esmolol* and Sodium Nitroprusside*<br>Nitroglycerin<br>Nicardipine*      | Labetalol* or Metoprolol*<br>Urapidil                  |
| <b>Eclampsia and severe preeclampsia</b> [15,24]                                               | Immediate reduction of SBP to < 160 mmHg and DBP < 105 mmHg.                                                                                                                                   | Labetalol* or Nicardipine*<br>Nifedipine, Urapidil<br>Magnesium sulphate | Consider delivery                                      |
| <b>Acute kidney injury</b> [1,2]                                                               | Decrease in avg. BP by 20-25%                                                                                                                                                                  | Labetalol*<br>Nicardipine*                                               | Clevidipine*<br>Urapidil                               |
| <b>Adrenergic crisis</b> [47]                                                                  | Decrease in avg. BP by 20-25%                                                                                                                                                                  | Phentolamine*                                                            | Nicardipine*<br>Clevidipine*<br>Labetalol*<br>Urapidil |

**Note:** \* – not registered in the Kyrgyz Republic; † – alternative approach is to use oral ACE inhibitors; however, titration of these drugs should begin with very low doses to prevent a sharp decrease in BP. Abbreviations: BP – blood pressure, AHP – antihypertensive drugs, SBP – systolic blood pressure, DBP – diastolic blood pressure, ARF – acute renal failure, HR – heart rate

**Source:** developed by the authors based on clinical guidelines and research

In most cases, with HC, it is recommended to ensure a rapid, but not more than 25% of the initial values, reduction in BP in the first hours of admission to the hospital [1]. The question of the degree and speed of further reduction in BP is decided individually in each case. The fastest reduction in BP should be carried out with a delaminating aortic aneurysm (by 25% of the initial level in 5-10 minutes, the optimal time to reach the target BP level of less than 120 mmHg is no more than 20 minutes) [34], and with severe acute left ventricular insufficiency [1]. For the treatment of HC, the following parenteral AHPs are used [1-2,8]. Vasodilators include nitroglycerin (preferred for ACS and acute left ventricular failure) and sodium nitroprusside (the drug of choice for acute hypertensive encephalopathy). Angiotensin converting enzyme (ACE) inhibitors include enalaprilate, which is preferred in acute left ventricular failure, but is contraindicated in pregnant women. Beta-blockers include metoprolol, esmolol, and labetalol (preferred for aortic dissociative aneurysm and ACS). Diuretics (furosemide for acute left ventricular failure), alpha-blockers (urapidil, phentolamine), calcium channel blockers (nicardipine, clevidipine), dopaminomimetic phenoldopam and antipsychotics (droperidol) are also used.

A.J. Peixoto [24] noted that most highly effective parenteral drugs (sodium nitroprusside, esmolol, labetalol, phentolamine, nicardipine, clevidipine, phenoldopam) are currently not registered in the Kyrgyz Republic. As for short-acting oral drugs, their use for the relief of HA is not indicated due to the danger of uncontrolled decrease in blood pressure. Short-acting nifedipine should not be used (the exception is the need for pre-hospital care for pregnant women with severe hypertension, but in this case, sublingual administration of short-acting nifedipine is not recommended). It is also important to note that magnesium sulphate is not an antihypertensive agent, its use for the relief of HC is not substantiated. The use of this drug is substantiated only in the presence of convulsive syndrome (including in pregnant women for the treatment and prevention of eclampsia) as anticonvulsant therapy, and for the treatment of hypomagnesemia in severe rhythm disorders. In most patients with HC, the results of examination for acute TOD are negative, and in this group of patients, BP reduction can be performed on an outpatient basis using AHP in accordance with the standards of the hypertension treatment algorithm with enhanced monitoring of adherence to therapy [1,6]. The therapeutic goal is a controlled reduction in BP to safe levels without the risk of hypotension [3,8].

### **Features of blood pressure control in inpatient and outpatient settings**

**Tactics for symptomatic increase in BP.** When registering high BP figures accompanied by nonspecific complaints without obvious markers of acute TOD, a watchful

waiting strategy is most appropriate. A randomised study in patients with low risk of HC showed that a two-hour period of rest demonstrated the same effectiveness for lowering BP as the appointment of antihypertensive therapy [48]. According to T.S. Anderson *et al.* [49], the administration of sedatives (including anxiolytics) also helps to normalise blood pressure if the leading trigger of the crisis was psychoemotional stress.

In some cases of marked symptomatic blood pressure elevation (but without typical signs of acute target organ damage), the treating physician or emergency medical specialist may, at their discretion, prescribe orally administered intermediate-acting antihypertensive agents (sustained-release nifedipine, moxonidine, carvedilol) [6]. The choice of the drug is made depending on the severity of the increase in BP and clinical symptoms. The rate of decrease in blood pressure should not exceed 25% in the first 2 hours, followed by reaching the target BP within a few hours (no more than 24-48 hours) from the beginning of therapy. Medications should be taken per os, sublingual administration is contraindicated due to the danger of a sharp uncontrolled decrease in BP. Gradual and stepwise titration of the recommended oral AHPs is recommended: after making a decision to introduce the drug, a follow-up period of at least 2 hours is proposed to assess the effectiveness and safety of lowering BP [1]. Further treatment may include the resumption or intensification of previous antihypertensive therapy or the appointment of new AHPs according to the acute hypertension treatment algorithm. In all cases, long-term control of BP is of paramount importance to prevent recurrent hypertensive crises and improve the overall prognosis [1,3].

**Tactics for an asymptomatic increase in BP.** It is necessary to focus separately on the tactics of treating patients with an asymptomatic increase in BP: this category does not belong to the HC. As a rule, these are patients with hypertension who have uncontrolled AH. Hypertension is often an asymptomatic disease, and even with significant increases in BP, clinical symptoms may be absent [3]. G.S. Stergiou *et al.* [27] argued that a single asymptomatic increase in BP in these patients should not be considered as an indicator of clinical deterioration and does not require immediate response, hospitalisation and emergency reduction in BP are not indicated. In this case, measures are needed to achieve effective AH control in these patients, that is, they should initiate or intensify long-acting oral AHPs according to the current algorithm [3,8,13]. The strategy of "rapidly lowering high BP" in asymptomatic patients using fast- and short-acting oral AHPs on an "as-needed" basis, as a reaction to incidental or routine BP measurements by a physician and/or self-measurement by the patient, is incorrect [27].

According to G. Mancia *et al.* [3], doctors should not recommend self-administration of fast-acting oral AHP with an increase in BP as an "emergency treatment".

This approach is useless, and sometimes even harmful to the patient, as it leads to increased variability in BP. It should be noted that increased BP variability is a poor prognostic sign for the development of cardiovascular complications, especially ACVA. In the treatment of AH, there is no goal to immediately reduce asymptomatic/low-symptomatic elevated BP. The goal is to gradually reduce it and keep it in the target range for the long term. The “time window” to achieve the target BP is 3-6 months with a titration step of 2-4 weeks. The strategy of antihypertensive therapy for hypertension is described in detail in the relevant clinical guidelines of the Ministry of Health of the Kyrgyz Republic [13].

**Treatment of acute hypertension in inpatient setting.** When patients are hospitalised, especially in emergency departments, there may be an acute increase in BP, which is not always associated with HC. Causes of blood pressure elevation in hospital settings are diverse and include common factors such as pain, anxiety, poor sleep, excessive fluid intake, and treatment-related factors (iatrogenic causes) [3,27]. Strategies for the treatment of elevated BP in inpatient patients ranged from the use of short-acting oral medications “as needed” to gradual titration of long-acting oral AHPs and intensive therapy with intravenous AHPs. The results of the conducted studies indicate the potential harm associated with intensive treatment with intravenous AHP, and with the use of drugs “as needed” to control blood pressure spikes in inpatient patients in the absence of HC.

G.S. Stergiou *et al.* [27] recommended avoiding this practice. A retrospective study of 66,140 patients hospitalised with non-cardiovascular diseases showed that intravenous therapy led to serious adverse outcomes, such as acute kidney injury and transfer to the intensive care unit. In this study, SBP was >180 mmHg in approximately 15% of patients [50]. B. Chaganti & R.A. Lange [51] conducted a similar observational study analysing data from almost 23,000 hospitalised patients. The study showed that patients treated for “blood pressure spikes” had higher rates of acute kidney injury and myocardial infarction. The optimal approach to the treatment of patients with acute elevation of BP in the hospital is the gradual and phased titration of the recommended oral AHPs. Intravenous treatment is indicated only in rare cases (if indicated at all). Adequate pain analgesia (whether it is postoperative pain or pain associated with other causes) often helps to stabilise BP even without the appointment of AHP [52-54]. In addition, measures to eliminate anxiety are recommended, and AHP withdrawal syndrome should be avoided [27,55]. Notably, even after intensive HC therapy in the hospital, most patients will continue to have hypertension on an outpatient basis, which requires the development of a coordinated strategy over several weeks or months to gradually achieve blood pressure control and prevent long-term complications.

**Features of blood pressure control in elderly patients.** Elderly patients with cardiovascular diseases, including hypertension, exhibit some characteristics that may differ from the general population, with a corresponding effect on diagnosis, treatment, and prognosis. This population represents a heterogeneous group, ranging from robust, independent individuals without comorbidities to highly dependent individuals. Therefore, an individual approach to variables beyond age is important for guiding goals and strategies for the prevention of cardiovascular diseases [56]. Based on the data of the world literature on the prevalence of geriatric syndromes in patients with arterial hypertension and the latest findings of M. Murzaibragimova *et al.* [57] in Kyrgyzstan, with a relatively high prevalence (40.1%) of cognitive impairment in patients with hypertension, it was decided to focus separately on blood pressure control in elderly and senile patients in a subsequent publication.

## Conclusions

Current diagnosis tactics for HC should be based not on absolute BP values, but solely on the facts of verified acute TOD. This diagnostic approach should become fundamental in the routine clinical practice of the Kyrgyz Republic. The term “uncomplicated hypertensive crisis” is outdated and excluded from international standards. A significant asymptomatic increase in BP in patients dictates the need for a planned, controlled intensification of basic antihypertensive therapy, rather than unjustified emergency medical interventions.

The diagnostic algorithm for initial contact with a patient should focus on the rapid search for “red flags” and confirmation of acute TOD using a regulated standard of clinical, laboratory, and instrumental methods. Management of true hypertensive crisis requires emergency admission to an intensive care unit and a strictly individualised approach. The choice of a priority parenteral drug and the rate of haemodynamic relief (lowering blood pressure) are strictly determined by the type of acute TOD that has developed. The routine use of short-acting nifedipine or magnesium sulphate for the relief of crisis conditions without specific indications has been recognised as pathophysiologically unjustified and poses a direct threat to the development of iatrogenic hypoperfusion of vital organs. The systematic development and implementation of unified standards for the management of hypertensive crises, based on the principles of evidence-based medicine, would enable the standardisation of clinical practice, improve patient safety in both infectious and therapeutic dimensions, and enhance long-term patient outcomes in the Kyrgyz Republic. For successful integration of updated algorithms, continuous educational programmes for primary care physicians, cardiologists, and emergency medical specialists are of critical importance. The purpose of such programmes is to overcome outdated

therapeutic stereotypes and to form alertness about the underdiagnosis of latent forms of TOD.

The shortage of emergency antihypertensive drugs remains a key barrier to the full implementation of international protocols in the Kyrgyz Republic. A priority step at the level of relevant departments of the Ministry of Health should be to resolve the issue of state registration and provision of hospitals with first-line parenteral drugs (labetalol, nicardipine, esmolol). Future research perspectives in this area include the initiation of large-scale cross-sectional epidemiological studies. An objective assessment of the real-world prevalence of different clinical phenotypes of acute TOD in the region

is required, including the establishment of local disease registries to enable continuous monitoring of clinical effectiveness and the pharmacoeconomic substantiation of implemented emergency care algorithms.

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## Conflict of Interest

None.

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